AMENDMENTS TO THE CLAIMS

This listing of claims will replace all prior versions and listings of claims in the application:

1. (Previously Presented) A method of treating a subject diagnosed as having a lysosomal storage disease selected from the group consisting of Fabry disease, Niemann-Pick disease, Pompe disease, and Gaucher disease, comprising first administering a gene therapy vector encoding a lysosomal hydrolase under the control of at least one tissue-specific regulatory element and then administering an exogenously produced natural or recombinant lysosomal hydrolase, such that the lysosomal storage disease is treated,

wherein:

the gene therapy vector is an adeno-associated virus (AAV), ~~and~~

the tissue specific regulatory element is a liver-specific regulatory element, and the lysosomal hydrolase is one that is deficient in the subject.

2. (Cancelled)

3. (Previously Presented) The method of claim 1, where the liver-specific regulatory element is chosen from at least one of a liver-specific promoter and a liver-specific enhancer.

4. (Original) The method of claim 1, where administering the gene therapy vector encoding a lysosomal hydrolase induces immunological tolerance to the lysosomal hydrolase.

5. (Cancelled)

6. (Previously Presented) The method of claim 1, where a lesser amount of the exogenously produced natural or recombinant lysosomal hydrolase is administered to the subject to treat the lysosomal storage disease than would be administered if the subject had not been administered a gene therapy vector encoding a lysosomal hydrolase or had been administered a gene therapy vector without a liver-specific regulatory element controlling expression of the lysosomal hydrolase.

7. (Original) The method of claim 1, where the lysosomal storage disease is Fabry disease.

8. (Currently Amended) The method of claim 7, where the treatment results in a decrease in globotriaosylceramide ~~globtriaosylceramide~~ (GL-3) in the subject compared to the GL-3 level in the subject before treatment.

9. (Original) The method of claim 7, where the lysosomal hydrolase is α-galactosidase A.

10. (Withdrawn) The method of claim 1, where the lysosomal storage disease is Pompe disease.

11. (Withdrawn) The method of claim 10, where the treatment results in a decrease in glycogen in the subject compared to the glycogen level in the subject before treatment.

12. (Withdrawn) The method of claim 10, where the lysosomal hydrolase is α-glucosidase.

13. (Cancelled)

14. (Previously Presented) The method of claim 1, where the gene therapy vector is chosen from adeno-associated virus 1 (AAV1), adeno-associated virus 2

(AAV2), adeno-associated virus 5 (AAV5), adeno-associated virus 7 (AAV7), and adeno-associated virus 8 (AAV8).

15. (Previously Presented) The method of claim 1, where the liver-specific regulatory element is a liver-specific promoter.

16. (Previously Presented) The method of claim 15, where the liver-specific promoter is a human serum albumin promoter.

17. (Previously Presented) The method of claim 1, where the liver-specific regulatory element is a liver-specific enhancer.

18. (Previously Presented) The method of claim 17, where the liver-specific enhancer is a human prothrombin enhancer.

19. (Cancelled)

20. (Previously Presented) A method of treating a subject diagnosed as having Fabry disease comprising first administering a gene therapy vector encoding α-galactosidase A under the control of a human albumin promoter and 2 copies of a human prothrombin enhancer and then administering an exogenously produced natural or recombinant α-galactosidase A, such that the Fabry disease is treated,

wherein the gene therapy vector is an adeno-associated virus (AAV).

21. (Cancelled)

22. (Withdrawn – Currently Amended) A method of treating a subject diagnosed as having Pompe disease comprising first administering a gene therapy vector encoding α-glucosidase under the control of a liver-specific promoter and optionally, at least one copy of a liver-specific enhancer, and then administering an

exogenously produced natural or recombinant α-glucosidase, such that the Pompe disease is treated,

wherein the gene therapy vector is an adeno-associated virus (AAV).

23-35. (Cancelled)

36. (Previously Presented) The method of claim 20, where administering the gene therapy vector encoding α-galactosidase A induces immunological tolerance to the α-galactosidase A.

37. (Previously Presented) The method of claim 20, where a lesser amount of the exogenously produced natural or recombinant α-galactosidase A is administered to the subject to treat the Fabry disease than would be administered if the subject had not been administered a gene therapy vector encoding α-galactosidase A or had been administered a gene therapy vector without a human albumin promoter and 2 copies of a human prothrombin enhancer controlling expression of the α-galactosidase A.

38. (Currently Amended) The method of claim 20, where the treatment results in a decrease in globotriaosylceramide ~~globtriaosylceramide~~ (GL-3) in the subject compared to the GL-3 level in the subject before treatment.

39. (Cancelled)

40. (Previously Presented) The method of claim 20, where the viral vector is chosen from adeno-associated virus 1 (AAV1), adeno-associated virus 2 (AAV2), adeno-associated virus 5 (AAV5), adeno-associated virus 7 (AAV7), and adeno-associated virus 8 (AAV8).

41. (Previously Presented) The method of claim 1, where the liver-specific regulatory element is DC190 (a human albumin promoter and 2 copies of a human prothrombin enhancer).

42. (Previously Presented) The method of claim 1, where the lysosomal storage disease is Niemann-Pick disease.

43. (Previously Presented) The method of claim 42, where the treatment results in a decrease in sphingomyelin in the subject compared to the sphingomyelin level in the subject before treatment.

44. (Previously Presented) The method of claim 42, where the lysosomal hydrolase is sphingomyelinase.

45. (Previously Presented) The method of claim 1, where the lysosomal storage disease is Gaucher disease.

46. (Previously Presented) The method of claim 45, where the treatment results in a decrease in glucocerebroside in the subject compared to the glucocerebroside level in the subject before treatment.

47. (Withdrawn) The method of claim 10, where the lysosomal hydrolase is glucocerebrosidase.